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EFFECTS OF ENVIRONMENT ON METABOLIC HEAT PRODUCTION, (U)

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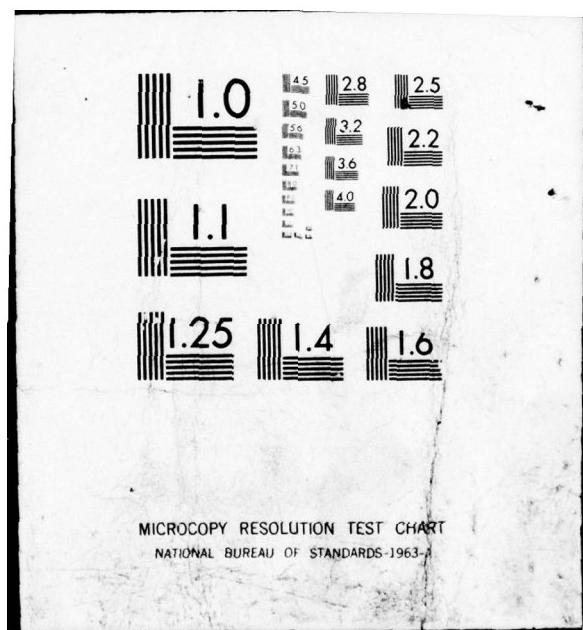
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About nervous system, 0.2 MET from the cardiovascular and respiratory systems and 0.5 MET from the remaining tissues comprising the lean body mass, at about 1.25 kcal/hr per kg of LBM, with muscle being a primary contributor. Fat is generally considered a negligible contributor to BMR, so changes in body fat may not alter BMR, but changes in muscle mass (i.e., increases as a result of increased work demands or decreases as a result of inactivity or inadequate nutrition) will; seasonal changes in BMR are frequently reported, but only occasionally attributed to altered physical activity or diet. Many factors alter resting metabolism in any environment: postural changes (5-10%), thermic effects of feeding (10-15%), altered deep body temperature (hyper/hypothermia) (~12%/ $^{\circ}$ C), but not heat loss per se, nervous tension (anxiety ~ 75%); in the cold, muscle tension increases metabolism (simple isometric contraction by up to 200% and shivering by up to 500%); sweating may or may not evoke increased metabolism, with any slight rise perhaps being masked by a decrease in the cost of maintaining vasoconstrictor tone. Working metabolic cost in any environment primarily appears to be independent of environmental temperature and a simple function of the square of the velocity of movement times the weight of the body plus clothing worn; in addition, with bulky cold weather clothing there is another 15% increase as a result of its "hobbling" impediment to movement. There are substantial effects of environmental terrain, with sand doubling and deep snow (20 cm of snow foot print depth) tripling the metabolic cost of walking at a given speed on a treadmill with a constant level of protective clothing. Finally, physical exhaustion from such high work loads, or incipient heat exhaustion from inability to eliminate heat being produced during work in cold or heat, or gained from a hot environment, can result in gross locomotor inefficiencies; energy costs can double from staggering or fighting to stay on ones feet. In summary, apart from: a) altered muscle mass and dietary influences, b) anxiety, altered posture or change in deep body temperature, c) muscle tensing or shivering and d) the mechanical effects of terrain, clothing weight and hobbling, or inefficient locomotion, we can conclude that ambient temperature per se (and acclimatization to it) does not appear to have any significant effect on metabolism per se.

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Effects of Environment on Metabolic Heat Production

by

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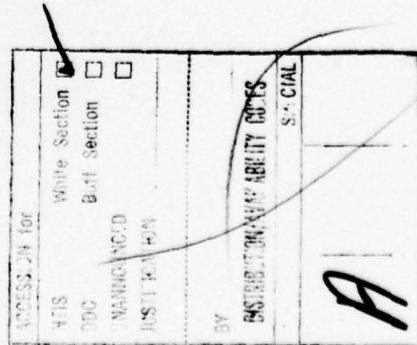
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Effect of Environment on Metabolism

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ABSTRACT

Three different states must be distinguished to look for effects of environment on metabolism: changes in the basal state should reflect effects of adaptation; changes at rest in the environment in question would reflect direct thermal effects; changes during work may reflect mechanical effects associated with the given environment. By definition, the basal metabolic rate in normal individuals should be independent of ambient environmental influences; contributions to the $\approx 40 \text{ kcal/m}^2 \cdot \text{hr}$ (0.8 MET) BMR include ~ 0.1 MET from the central nervous system, ~ 0.2 MET from the cardiovascular and respiratory systems and ~ 0.5 MET from the remaining tissues comprising the lean body mass, at about 1.25 kcal/hr per kg of LBM, with muscle being a primary contributor. Fat is generally considered a negligible contributor to BMR, so changes in body fat may not alter BMR, but changes in muscle mass (i.e. increases as a result of increased work demands or decreases as a result of inactivity or inadequate nutrition) will; seasonal changes in BMR are frequently reported, but only occasionally attributed to altered physical activity or diet. Many factors alter resting metabolism in any environment: postural changes (5-10%), thermic effects of feeding (10-15%), altered deep body temperature (hyper/hypothermia) ($\sim 12\%/\text{ }^\circ\text{C}$), but not heat loss per se, nervous tension (anxiety $\sim 75\%$); in the cold, muscle tension increases metabolism (simple isometric contraction by up to 200% and shivering by up to 500%); sweating may or may not evoke increased metabolism, with any slight rise perhaps being masked by a decrease in the cost of maintaining vasoconstrictor tone. Working metabolic cost in any environment primarily appears to be independent of environmental temperature and a simple function of the square of the velocity of movement times the weight of the body plus clothing worn; in addition, with bulky cold weather clothing there is another 15% increase as a result of its "hobbling" impediment to movement. There are substantial effects of environmental terrain, with sand doubling and deep snow (20 cm of snow foot print depth) tripling the metabolic cost of walking at a given speed on a treadmill with a constant level of protective clothing. Finally, physical exhaustion from such high work loads, or incipient heat exhaustion from inability to eliminate heat being produced during work in cold or heat, or gained from a hot environment, can result in gross locomotor inefficiencies; energy costs can double from staggering or fighting to stay on ones feet. In summary, apart from: a) altered muscle mass and dietary influences, b) anxiety, altered posture or change in deep body temperature, c) muscle tensing or shivering and d) the mechanical effects of terrain, clothing weight and hobbling, or inefficient locomotion, we can conclude that ambient temperature per se (and acclimatization to it) does not appear to have any significant effect on metabolism per se.



INTRODUCTION:

An extensive literature exists on the interaction between environment and metabolism; some studies suggest increases, others decreases and still others no changes in metabolism as a result of exposure to a given thermal environment. These conflicting results can sometimes be attributed to problems of experimental technique, subject anxiety, improper basis for comparison, flawed experimental design, etc., but frequently the problem is in the definition of an environmental effect.

Use of different techniques (or the same techniques by different investigators) for measurement of metabolic rate in otherwise comparable studies, or even within a single study because of difficulties of transporting laboratory personnel and equipment to the field, can lead to erroneous suggestions of small environmental effects on metabolism. Determinations of BMR in a large series of Korean subjects (34) gave a relatively "standard" value of $40 \text{ kcal/m}^2 \cdot \text{hr}$ using a Sanborn metabulator compared to $\sim 38 \text{ kcal/m}^2 \cdot \text{hr}$ using a Douglas bag and Scholander analysis technique. This 5% difference in BMR is comparable to the 4.4% found by Swift between the 24 hour energy expenditure assessed by direct and indirect calorimetry (as reported by Garrow (18)). While this demonstrates the reproducibility of metabolic rate determinations on groups of subjects, and demonstrates a validity for the indirect approach, it also documents the potential discrepancy between techniques under the best of laboratory conditions. Field estimates of the 24 hour activity regimen by subject diaries maintained on a minute to minute basis (42) or filled out every few hours (4, 14) are certainly better than end of day recall (35), but anything would be preferable to the too frequent statement that activity was comparable in both periods (or environments); however, environmental effects

on metabolism have been inferred from even less documented studies. Edholm (14) clearly demonstrated that energy expenditure of 12 military cadets all under the same living conditions and performing similar activities varied by about 100%, from ~ 2400 kcal/day to ~ 4800 kcal/day; in this same study, while food intake ranged between 2400 kcal and 5500 kcal it was not correlated with energy expenditure. One of the key papers suggesting an effect of environment on metabolism (32) was based on field surveys of food intake at military camps in hot, temperate and cold environments. However, subsequent studies (55) showed little difference in caloric intake across hot, temperate and cold environments, particularly if adjusted for body weight and the weight of clothing; a caloric intake of 47-49 kcal/kg. day was associated with moderate work in all three climates. Other workers have attempted to develop corrections in the laboratory for the weight of cold weather clothing worn in the field, but estimating the caloric cost of work in heavy arctic clothing using a bicycle ergometer (22) can lead to a serious underestimate of the caloric cost of work while walking in such clothing on a treadmill (52); furthermore, even walking on a treadmill with the complete 12.7 kg of U.S. Army uniform ensemble worn in the Arctic can give no hint on the cost of walking on snow.

Subject anxiety is seldom remarked upon in reports of metabolic studies, despite the finding (58) that with repeated measurements of well-trained subjects, the basal metabolic rate averages some 6% below the classic Mayo foundation standards (9). Garrow recently demonstrated (18) a near doubling of resting metabolic rate upon a "temporary anxiety" but minimal physical disturbance of a subject, with the return to the initial resting value delayed for about 45 minutes. Renbourne (personal communication) reported a marked, acute increase in oral temperature in a group of volunteers who viewed a short, purposely gory, movie of

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venipuncture just prior to their proposed donation of blood. It seems probable that the reports of very high resting metabolic rates in Eskimos (or other native populations) can be in part explained in terms of their response to an investigator approaching with nose clip, mouthpiece, valves, etc., as well as in terms of an environmental response or in terms of response to the 25 to 30% of the Eskimo dietary calories as protein (31,45).

Further confusion has been wrought, unwittingly, by the adoption of various bases to normalize metabolic rate for inter- or intra-individual differences. Height, weight, body surface area (by a variety of formulae), ponderal index, body mass index, and various estimates of lean body mass or fat free body weight have all been used. Durnin (13) concluded that there was no purpose in using surface area rather than body weight as a reference, based on a correlation of 0.92 between body weight and standing surface area and the correlations between energy expenditure while walking on a treadmill of 0.77 with body weight and 0.78 with body surface area. Other authors (44), in a study of such reference bases, reported no differences in correlation between oxygen consumption and weight, lean body mass, body surface area (by four different formulae) or body mass index (weight/height²); all gave r values between 0.61 and 0.65 which suggests that only about 40% of the metabolic rate (i.e. $r^2 \approx 0.4$) in this careful laboratory study could be related to these reference bases. In this same study, a crude estimate of lean body mass (LBM) gave about the same r value. Miller and Blyth (38) found BMR correlated as $r = 0.92$ with LBM, using an improved estimate of LBM, contrasted with 0.54 when correlated with surface area and 0.85 with weight; they found correlations of about 0.8 between LBM and both weight and surface area, and attributed much of the validity of the latter two for metabolic reference standards.

to their correlation with LBM. In correcting their caloric requirements across the different environments studied, Welch *et al.* (55) pointed out that use of body weight, *per se*, as a reference base implied a relationship that only existed during physical work; they used body weight^{0.7} as their unit of reference for resting metabolic rate following the concept of Galvao (17); in their study on variation in metabolism with changes in food, exercise and climate (4), the difference in resting metabolic rates between climates was not significant when this base was used.

Experimental designs which use different techniques in the different environments, or which involve groups of subjects who differ in their familiarity with the procedures, or which fail to balance order of environmental exposures using the same subjects, can obviously introduce serious errors from differences in anxiety level associated with unfamiliarity with technique, procedures or environments. Failure to balance designs to compensate for changes in body weight, whether induced by the differing work demands of the environment or by differences in diet associated with the environment, can also introduce errors.

A major source of difficulty in defining any influence of environment on metabolism is simply in the definition of an environmental effect. Failure to distinguish environmentally associated differences in body weight, activity patterns, physical demands, diet, restlessness, anxiety, clothing and/or loads carried, circadian effects and the like, can lead to the conclusion that there is an environmental effect of heat or cold *per se* on metabolism when, in fact, there is only an effect of one or more of these environmentally associated factors. As shown below, such imprecision in the definition of "environmental effects" on metabolism may be a major basis for the conclusion that such effects exist (15).

It seems appropriate to dissect any attempt to evaluate environmental

effects into three distinct areas of metabolic response: changes in basal metabolic rate, in a thermoneutral environment, should only reflect effects of adaptations by the body tissues in response to the various stresses of an environment; changes in resting metabolic rate, in the environment in question, should also reflect direct thermal effects on the body; changes in metabolic rate during work might well mask such adaptation and/or direct thermal effects, while revealing mechanical effects associated with the environment.

BASAL METABOLIC RATE

By definition, the basal metabolic rate is that of an individual in a neutral thermal environment, at least 12 hours post-prandial, at ease both mentally and physically. It is usually determined in the early morning, when it is lowest, preferably on first waking (or even still somnolent as in our studies) but, in any event, should be taken at the same time of day (1). If studies involve the rapid transfer of the subjects across time zones to new climates, there may be an additional problem since the period for circadian adjustment of BMR may or may not fit the 2 to 3 hours shift per day rule of thumb for resetting the general physiological responses to rapid time zone translocation; the $\sim 1^{\circ}\text{C}$ circadian change in deep body temperature at rest could produce a variation of $\sim 12\%$ in measured metabolic rate during the first few days following transport, if time of day (as sensed by the body) is not controlled.

Early studies suggested that, at rest, the central nervous system utilized about 46 ml of O_2 per minute, the kidneys about 14 ml, the liver about 35 ml and spleen and intestines perhaps 18 ml in the fasting state, bringing the total to about 113 ml O_2/min (2). This is half the usual standard BMR value of 250 ml O_2/min ($=72 \text{ kcal/hr or } 0.8 \text{ MET}$, where 1 MET is defined as a heat production of 50

$\text{kcal/m}^2 \text{ hr}$); the cardiovascular system (heart) and respiratory muscles require an additional 40 ml per minute at rest (1). Thus about 18% of the BMR is accounted for by the demands of the CNS, about 27% by the digestive and excretory systems, and about 16% by the circulatory and respiratory systems, leaving 39% for the remaining body tissues. Fat, perhaps erroneously, is generally considered a negligible contributor to the BMR, so most of this 39% reflects the demands of the remaining tissues comprising the lean body mass, with skeletal muscle being the primary contributor. Thus, as suggested by Rubner in 1902 (46), the tissues comprising the lean body mass are the primary source of basal metabolic demands, at a rate of about 1.25 kcal/hr per kg of lean body mass.

If we look at these separate components contributing to the BMR, there seems little likelihood that their demands in a thermoneutral environment in a fasting state are apt to be altered unless there is a significant change in their mass or in the basic control mechanisms that limit cell respiration and function. As shown in Figure 1, redrawn from data of Tzankoff and Norris (54), the contribution of non-muscle tissue to the total BMR is relatively constant throughout life, while the muscle tissue contribution falls by about 3.7% per decade of life (after ~30); in an earlier report (53), they showed that these age related decreases in BMR were totally accounted for by concurrent decreases in the mass of metabolically active, creatinine producing skeletal muscle. Thus, any alteration of muscle mass should produce a corresponding alteration in BMR. Differing environments can result in altered physical work demands, and the resultant, unplanned physical conditioning can alter muscle mass, with little notable weight-change as muscle tissue replaces fat. McDougall et al. (36) recently showed increases in arm circumference (~11%) and in muscle concentrations of creatine (~39%), ATP (~18%) and glycogen (~66%)

after five months of elbow extensor training, and decreases of ~5% in circumference and of ~40% in glycogen after 5 weeks of arm immobilization. As shown below, energy costs can double simply walking over sand, or can triple over snow with the added burden of heavy clothing: the increased muscle mass associated with the increased work demanded by living in extreme environments could therefore readily account for the early reports of a slightly increased BMR in man in the cold (7,28,51).

Altered regulation of metabolism at the cellular or subcellular level could also account for an increase in BMR. Current research suggests that heat production at the cellular level has three components: an essential heat associated with the continuous catabolism-anabolism cycles of body tissue turnover; an obligatory heat associated with various transport cycles (e.g., the Na pump); and, in homeotherms, regulatory heat required for temperature regulation. My interpretation of these fundamental mechanisms, based largely on the work of Girardier, Rossier, Seydoux and others (59) is presented in Figure 2. These multiple paths which link oxygen, substrate (e.g. glucose) and, chemical energy stores (e.g. ADP↔ATP) have varying efficiencies, with about 39% of the energy change storables for eventual use (and release as heat then) and about 61% appearing as heat directly.

While such mechanisms could change in response to environmental exposures, U.S. Army laboratory studies involving cold acclimatization of men in climatic chambers, showed no change in BMR for groups of men studied either at the end of the summer or at the end of the winter (11). Indeed, in another study of chamber cold acclimation (33), not only was BMR the same before and after the acclimation period, but resting oxygen uptake only increased by about 14% after acclimatization during infusion of norepinephrine; the drug produced an elevation in blood

pressures (with bradycardia) and the subjects were aware when infusion was started so that the increased vasomotor tone and/or associated anxiety could have accounted for some -- or all -- of this slight elevation. Brown fat, a major site for nonshivering thermogenesis in response to norepinephrine modulation of sodium transport and intracellular pH as suggested by Chinet (59), has not been reported in man. Although Rubner, as early as 1902 (46), suggested that man exhibited chemical, i.e. non-shivering heat production, temperature regulating mechanisms, it seems reasonable to suggest that it is primarily the modifications in peripheral blood flow which enhance comfort following cold acclimation (60). Even in small Arctic mammals, acclimatization to winter does not usually elevate resting metabolic rate in a comfortable environment (26). Thus, despite the older view that BMR is negatively correlated with average temperature -- with each $^{\circ}$ F from a mean 70° F base assumed to change BMR by 4 kcal -- it seems more reasonable to conclude that there is no meaningful effect of cold on BMR, and that studies suggesting such changes have reflected alteration in diet (which can change BMR (5,45)) or in lean body mass in response to the activities demanded by the environment.

There are studies suggesting decreased BMR in response to living in the heat but, as suggested in Figure 2, little reason to anticipate any as long as the BMR is measured in a truly thermoneutral environment and there is no change in active body mass as a result of altered activity level, no change in diet (5), and no change in endocrine levels.

RESTING METABOLIC RATE:

Resting metabolic rate in a thermoneutral environment should be expected to be essentially independent of environmental influences, except for those effects

cited above for BMR; changes in active tissue mass would also alter resting metabolic rate and the nature of the diet and the time since the last meal can substantially alter resting metabolic heat production (4,5,18). Scholander *et al.* have suggested that the "critical air temperature", (i.e. for comfort and to avoid shivering), which for a naked man is 27-29°C, is the same for peoples whose evolution has occurred in contrasting climates (47), and other studies (4) have shown no difference in resting metabolic rate across desert, temperate and arctic environments. Postural changes induced by exposure to a hot or cold environment, with people at rest in the cold tending to curl up (foetal position) to minimize the exposed surface area and at rest in the heat to "spread eagle" to expose as much surface as possible to the environment, can alter heat production at rest by some three to five percent. In general, however, resting metabolic rate of nude and/or clothed men is unaffected by environments from 22-35°C during the first hours (16,25,57). A non-thermoneutral temperature can, however, have a much more substantial effect on resting metabolic rate than simply this postural change. If deep body temperature is altered, there is a corresponding alteration in metabolic rate (50); an increase in body temperature of 1°C, whether the result of febrile disease or body heat storage at high ambient temperatures, increases metabolic rate by about 12% (12) as a simple result of the Arrhenius logarithmic increase in the velocity constants of some of the mechanisms shown in Figure 2.

In the cold, the generalized increase in muscle tone that precedes shivering (6), can substantially increase oxygen consumption. Also, voluntary isometric contractions can increase heat production; an isometric contraction which subjectively simulated the tension sensed in the cold, increased heat production by 36% (51). Shivering, which begins locally and only gradually recruits involvement

of the entire body, can increase "resting" heat production to a 425 kcal/hr level (30,51). However, shivering is not a successful defense against cooling for a nude man; shivering is generally preceded by pilo-erection, usually remarked as "goose flesh", in a vestigial attempt both to increase the thickness of the external air layer and to protect it from being decreased by the body motion which accompanies shivering. In the absence of a fur coat, such pilo-erection is ineffective in preserving or increasing the thickness of the insulating trapped air layer which surrounds an inactive individual and, since shivering also effectively disrupts the vasoconstrictive insulation between the body's skin surface and its heat producing core, an individual (particularly one with a thick subcutaneous fat insulative layer) who shivers may increase his heat loss to a greater extent than his heat production.

The stimulus for shivering usually originates as a function of mean skin temperature (\bar{T}_s), but the rate of change of skin temperature may be as important as its absolute level. Shivering is also modulated by deep body temperature (T_{re}) levels; ingestion of one liter of ice water shortened the time to onset of shivering by six minutes in one study (10). Hayward *et al.* (27) have recently suggested that the steady-state metabolic rate (M in W/kg) of a non-exercising man could be predicted as:

$$M = .0314 (\bar{T}_s - 42.2^\circ\text{C})(T_{re} - 41.4^\circ\text{C})$$

where the equation is "limited by the low level of BMR (1.1 W/kg) and by the high level of peak metabolic rate (6.4 W/kg)"; the kg reference is to the total body weight rather than the metabolically active lean body mass.

There are considerable inter-individual and inter-ethnic group differences in the metabolic response to cold exposure. Hardy and Du Bois (24) found an earlier metabolic increase on cold exposure of women than men, and there are substantial

differences in body cooling and heat production as a function of subcutaneous fat thickness (3,60), as a function of acclimation or acclimatization (48) and as a result of the adaptive responses of various ethnic groups (47,58). Although the latter frequently reflect enurement to discomfort or clever use of behavioral mechanisms like sleeping next to a fire (61) or sleeping with a narcotizing leaf under the tongue, two different generalized types of response have been suggested: a metabolic response, with increased heat production (48) and an insulative response, primarily involving endurance of a greater fall in skin temperature (49).

A small effect on resting metabolism in the heat is frequently reported; Pitts (43) reported an increase of up to 7 kcal/hr while standing, and most authors find small decreases at rest in the heat (56). The changes reported appear compatible with altered circulatory demands; altered vasomotor tone and perhaps a diversion of blood from the muscle and other deep tissues to the skin. An accompanying increase in anaerobic metabolism, as suggested by Williams *et al.* (56), could explain the changes in resting metabolism in the heat but, while changes in excess lactate (29) can be measured for a reasonable level of work in comfortable, in contrast with hot conditions, the magnitude of change in metabolism at rest in the heat is generally too small to expect to detect changes in the circulating levels of lactate and pyruvate.

In addition to the circulatory effects of heat, if hyperventilation occurs as a response to the heat (21), then some increase in the metabolic cost of respiration would be incurred, particularly since respiratory work is only about 5% efficient (39); however, this is probably already included as a part of the 12% increase in metabolism per °C rise in body temperature since hyperventilation in the heat occurs in company with elevated deep body temperatures (8,21).

WORKING METABOLIC RATE:

Turning finally to the consideration of environmental effects during work, there are many "environmental" factors which alter the mechanical cost of physical work. We (19,41) have demonstrated that the metabolic cost of walking (M in watts) can be formulated as:

$$M = 1.5W + 2.0 \frac{L+W}{L/W}^2 + \frac{L+W}{L/W} 1.5V^2 + 0.35 VG$$

where: W = body weight (kg)

L = load weight (kg)

V = velocity (m/s)

G = grade (%)

= terrain coefficient

If one considers the weight of protective clothing in the cold (e.g. the 12.7 kg worn by a U.S. soldier in the Arctic), and also the effective five fold cost of the weight of footwear (1 kg on the foot = 5 kg on the back (40)) plus the increase in metabolic cost as a result of the "hobbling" of the clothing, which amounts to about 15% in addition to the cost incurred by weight of such bulky clothing (52), it is clear that the increased work demanded represents a significantly increased metabolic cost, as well as a likely source of stimulus to increased lean body mass.

The terrain coefficient () in the above formula is the empirical ratio between the measured metabolic cost at a given speed with a given load walking on a treadmill and the measured cost on the terrain in question. For hard packed snow, the energy cost is increased by only 30% (i.e. $\alpha = 1.3$) but, with increasing depth of footprint in soft snow, the terrain coefficient is greater: $\alpha = 1.3 + 0.08 \times \text{cm footprint depth}$ (41). The cost of walking in deep, soft snow becomes so enormous, in fact, that the velocity must be reduced to avoid exhaustion, to a level

where the metabolic demand is about 500 Watts; this level represents the "voluntary, sustained hard work level" of our average military subject (41). Walking on soft sand ($= 2.1$) or in jungle terrain (heavy brush $= 1.6$ and swamp $= 1.8$) is not quite as demanding as deep, soft snow, but there still can be potent "environmental" demands on metabolism in a hot environment.

The excessive work demanded by operations in desert, jungle and arctic environments, coupled with the need for protective clothing (against sun, insects and abrasion in the heat, as well as in cold) frequently result in severely elevated body temperatures in arctic as well as tropic environments; with incipient heat exhaustion, metabolic costs climb dramatically just to stay on one's feet (20).

Apart from these major, mechanical "environmental" influences on metabolic cost, it seems clear that there is little effect of environmental temperature per se on working metabolic costs. Indeed, in a Norwegian study of the effects of cold on men exposed during two months above treeline with inadequate clothing, the investigators point out that "in the daytime the men could exercise all they wanted and therefore the problem of keeping warm pertained largely to the night time when they were at rest and asleep" (48). No differences were found in metabolic costs during fixed exercise in the cold, before or after the long cold acclimating exposure; the authors conclude that "neither during exercise nor during rest in the cold did the acclimated men save on calories by insulating themselves with a cold vasoconstricted shell". Similar conclusions on the lack of regulation of heat loss were drawn in an earlier study by this same group (47). However, Wyndham et al. (62) suggest that the important mechanisms of man's long term adaptation to cold include both a gain in weight (but they did not determine whether the gain was in subcutaneous fat or in metabolically active, lean body mass) and an improved tissue

insulation.

Wyndham's group (63), also studied oxygen uptake in hot-wet environments, and found no effect of heat or of heat acclimatization on working oxygen consumption nor even on maximum oxygen consumption in the heat, although in an earlier study (56) they had reported that oxygen consumption at submaximal work rates were significantly lower in the heat; the originally reported differences were small, being on the order of 1 kcal/min. In our own studies (23), we found a significant difference in working metabolic rate between a hot-wet and a hot-dry condition, but the actual difference was negligible, 321 kcal/hr in the hot-wet as opposed to 314 kcal in the hot-dry condition; these values compare to a 303 kcal/hr value predicted ignoring any rise in deep body temperature. We agree with Malhotra et al. (37), as shown in Figure 3, that when metabolic costs during work are properly expressed per kg of total body, load and clothing weight there are no practical, and probably few reproducible differences in metabolic demands during work in hot or cold environments.

CONCLUSION:

Only a small portion of the massive literature looking for environmental thermal effects on metabolism has been presented. The literature is full of conflicting reports; however, it seems safe to conclude that, in the absence of altered diet or lean body mass, if there is no direct cold-induced stimulation of muscle tone or shivering and if clothing weight and terrain effects are properly accounted for, there is little meaningful effect of environment on metabolism.

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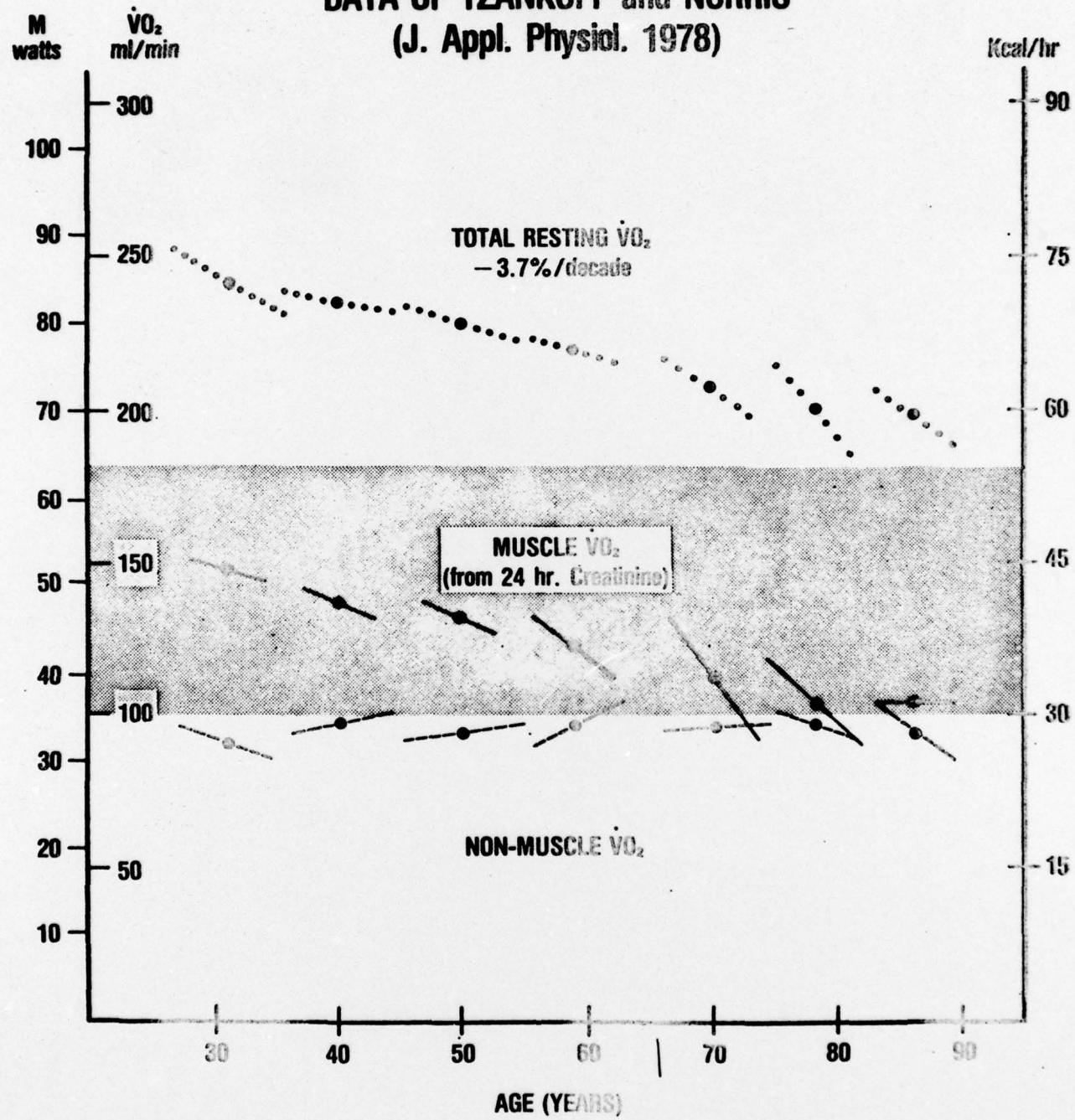
FIGURE CAPTIONS

Figure 1. Change in resting metabolic rate with age, and the relative contribution of non-muscle and muscle tissues to metabolism (from data of Tzankoff and Norris (53,54)).

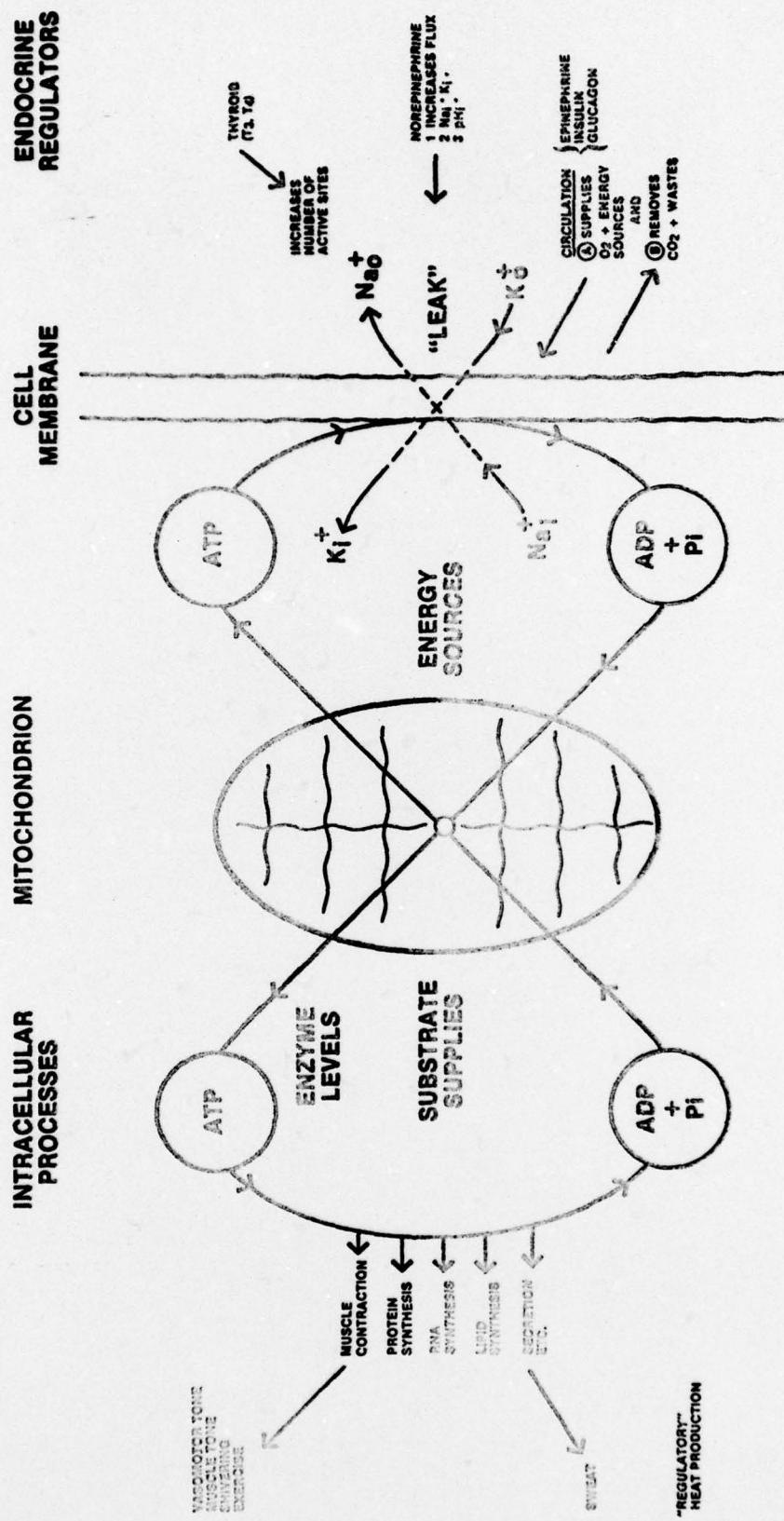
Figure 2. Elements in metabolic heat production and its regulation at the cellular level (from a variety of sources including (59)).

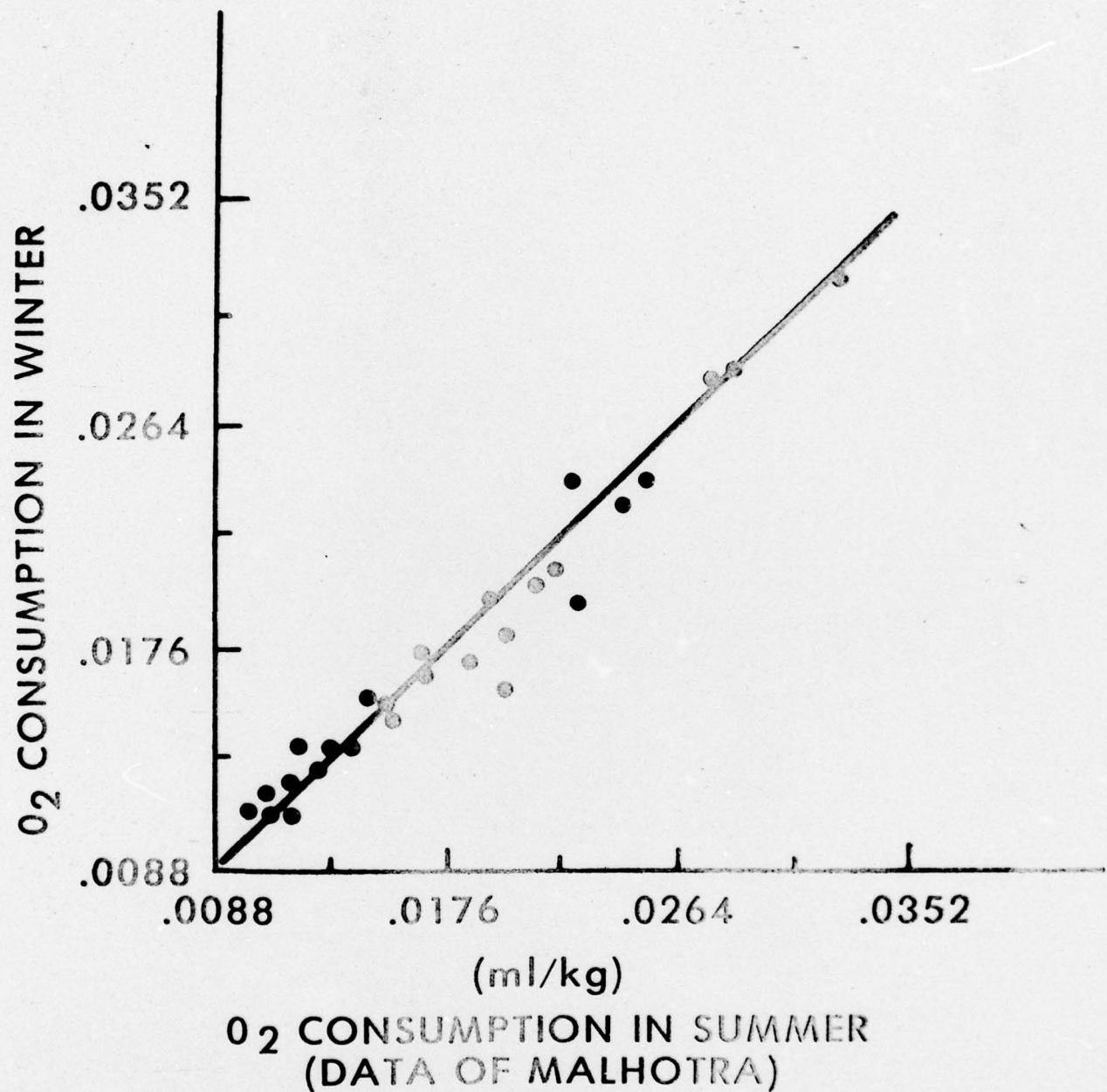
Figure 3. Working and resting oxygen consumption in summer and winter for a variety of activity levels (from Malhotra (37)).

DATA OF TZANKOFF and NORRIS
(J. Appl. Physiol. 1978)



NATURE OF BIOCHEMICAL PATHWAYS FOR HEAT PRODUCTION
 (Expanded from diagram of Rossier)





O_2 CONSUMPTION IN SUMMER
(DATA OF MALHOTRA)